

## Modulation of wireless (2.45 GHz)-induced oxidative toxicity in laryngotracheal mucosa of rat by melatonin

Giray Aynali · Mustafa Nazıroğlu · Ömer Çelik ·  
Mustafa Doğan · Murat Yarıktaş · Hasan Yasan

Received: 21 January 2013 / Accepted: 27 February 2013 / Published online: 12 March 2013  
© Springer-Verlag Berlin Heidelberg 2013

**Abstract** It is well known that oxidative stress induces larynx cancer, although antioxidants induce modulator role on etiology of the cancer. It is well known that electromagnetic radiation (EMR) induces oxidative stress in different cell systems. The aim of this study was to investigate the possible protective role of melatonin on oxidative stress induced by Wi-Fi (2.45 GHz) EMR in laryngotracheal mucosa of rat. For this purpose, 32 male rats were equally categorized into four groups, namely controls, sham controls, EMR-exposed rats, EMR-exposed rats treated with melatonin at a dose of 10 mg/kg/day. Except for the controls and sham controls, the animals were exposed to 2.45 GHz radiation during 60 min/day for 28 days. The lipid peroxidation levels were significantly ( $p < 0.05$ ) higher in the radiation-exposed groups than in the control and sham control groups. The lipid peroxidation level in the irradiated animals treated with melatonin was significantly ( $p < 0.01$ ) lower than in those that were only exposed to Wi-Fi radiation. The activity of glutathione

peroxidase was lower in the irradiated-only group relative to control and sham control groups but its activity was significantly ( $p < 0.05$ ) increased in the groups treated with melatonin. The reduced glutathione levels in the mucosa of rat did not change in the four groups. There is an apparent protective effect of melatonin on the Wi-Fi-induced oxidative stress in the laryngotracheal mucosa of rats by inhibition of free radical formation and support of the glutathione peroxidase antioxidant system.

**Keywords** Melatonin · Larynx · Trachea · Oxidative stress · Wireless devices

### Introduction

Wireless devices usages in industrial, scientific, medical, military and domestic applications, with potential leakage, of such radiation into the environment have increased by leaps and bounds in past decade [1]. From being a luxury and limited to the wealthy, wireless devices especially near 2.45 GHz is indispensable in daily lives [2]. However, every technological advance and its overuse possess possible adverse effects [3].

Exposure to electromagnetic radiation (EMR) induces degenerative effects via two ways, namely directly or indirectly. Direct effects of EMR induce production of reactive oxygen species (ROS), including superoxide anion, hydrogen peroxide, and hydroxyl radicals. The ROS contribute to tissue and DNA damages [1]. Exposure to 2.45 GHz EMR causes an increase in lipid peroxidation levels and a decrease in the activity of enzymes that prevent or protect against lipid peroxidation in tissues [4, 5]. The human cells have nonenzymatic and enzymatic antioxidant systems against degenerative effects of ROS. Glutathione

G. Aynali · M. Yarıktaş · H. Yasan  
Ear, Nose and Throat, Head and Neck Surgery Department,  
School of Medicine, Suleyman Demirel University,  
Isparta, Turkey

G. Aynali (✉)  
Modernevler Mah. Çevre Yolu 142. Cad. No. 7 İksir  
Apt D :7, 32200 Isparta, Turkey  
e-mail: giraynali@yahoo.com; giraynali@med.sdu.edu.tr

M. Nazıroğlu · Ö. Çelik  
Biophysics Department, School of Medicine, Suleyman  
Demirel University, Isparta, Turkey

M. Doğan  
Ear, Nose and Throat, Head and Neck Surgery Department,  
Isparta State Hospital, Isparta, Turkey

(GSH) is the most abundant thiol antioxidant in mammalian cells [6]. The GSH is an endogenous tripeptide that acts both as a nucleophilic scavenger of numerous compounds and as a substrate in the selenium-dependent GSH peroxidase (GSH-Px)-mediated destruction of hydroperoxides [7]. Recently, we observed proliferative effects of 2.45 GHz EMR on human leukemia cancer cells through the over production of ROS and  $\text{Ca}^{2+}$  influx [8]. Melatonin, the main secretory neurohormone of the pineal gland, has been considered a potent antioxidant that detoxifies a variety of ROS in many pathophysiological states [9]. Melatonin also plays a significant role in cancer production against a variety of cancer diseases including larynx cancer whose pathogenesis involves damage of ROS [10]. Recently, we observed also modulator role of melatonin on 2.45 GHz-induced oxidative stress in rat dorsal root ganglion neurons [11]. To our knowledge, there is no report on 2.45 GHz-induced oxidative stress in laryngotracheal mucosa of human and experimental animals. Melatonin may modulate Wi-Fi (2.45 GHz)-induced oxidative stress in laryngotracheal mucosa of rats and it needs to be clarified.

Larynx carcinoma comprises 25 % all head and neck cancers and 2–3 % malignant tumors. Several reports studying the oxidative related values in larynx cancer indicated the importance of oxidative stress. Recently, Karaman et al. [12] reported that the oxidant/antioxidant balance was impaired in favor of lipid peroxidation and DNA damage in patients with laryngeal squamous cell carcinoma. Manjunath et al. [13] investigated the level of oxidative stress in laryngeal and hypopharyngeal cancer patients in a prospective study and they observed positive correlations in oxidative stress and initiation of laryngeal and hypopharyngeal cancers.

The purpose of the present study was to investigate the effects of 2.45 GHz EMR on oxidant–antioxidant changes in laryngotracheal mucosa of rat and the possible protective effects of melatonin.

## Materials and methods

### Chemicals

All chemicals were of analytical grade, obtained from Sigma-Aldrich Chemical Inc. (St. Louis, MO, USA) and all organic solvents from Merck Chemical Inc. (Istanbul, Turkey). All solutions, except phosphate buffers, were prepared daily and stored at 4.0 °C. The reagents were allowed to equilibrate at room temperature for at least 30 min before analysis. The phosphate buffers were stable at 4.0 °C for 1 month.

### Animals

This study is planned and organized as completely double-blind. All experimental procedures had been approved by Medical Faculty Experimentation Ethics Committee of Süleyman Demirel University (Protocol Number; 2011-01/02). Male Wistar albino ( $n = 32$ ) rats were used in the current experiment. At the start of the experiment, the rats were 16 weeks old and weighed 220–240 g. Animals were maintained and used in accordance with the Animal Welfare Act and the Guide for the Care and Use of Laboratory animals prepared by the Suleyman Demirel University. The rats were housed individually in stainless steel cages in a pathogen-free environment in our laboratory at  $22 \text{ }^{\circ}\text{C} \pm 3$  with light from 0800 to 2000 hours and allowed free access to water and fed a commercial diet. Environmental average light intensity was 4,000 lux and humidity was  $40 \pm 10 \%$ .

### Study groups

After 1 week adaptation process, the animals were randomly categorized into four equal groups, namely cage control rats, sham control rats, rats exposed to 2.45 GHz during 60 min/day for 30 days and same as the previous group, 2.45 GHz-exposed rats treated with intraperitoneal (ip) injections of melatonin at a dose of 10 mg/kg/day.

The 1-h exposure to irradiation in last two groups took place between 9 AM and noon each day. The first dose of melatonin administration was performed 24 h prior to exposure. Sham control rats received ip injections of isotonic saline solution at an equal volume to that of melatonin used in the last melatonin group.

Melatonin was dissolved in a small (100  $\mu\text{l}$ ) amount of dimethyl sulfoxide and then diluted with physiological saline solution. The volume of melatonin solution injected daily was 0.1 ml. The melatonin dose used in this study was chosen on the basis of our previously published experiment [11].

### Exposure system and design

Details of exposure system have been described in detail elsewhere [4]. A “SET ELECO” generator from Set Electronic Co., Istanbul (Turkey), provided with a half-wave dipole antenna system was used to irradiate the cells with a 2.45 GHz radio frequency with 217 Hz pulses. The electric field density was set at 11 V/m to get a 0.1-W/kg whole-body average specific absorption rate (SAR).

Radiation reflection and exposure were measured with a Portable radio frequency Survey System (HOLADAY, HI-4417, Minnesota, USA) with a standard probe. The electromagnetic radiation dose was calculated from the

measured electric field density (V/m). SAR values were calculated using electrical properties of tissue sample and measured electric field intensities for every distance in certain frequency. These values are shown in Fig. 1.

We used eight rats in the exposure system at the same time (Fig. 1). This device is organized with a special cylindrical strainer which is appropriate for exposure condition and physical size of one rat (length 15 cm, diameter 5 cm). The noses of the rats were positioned in close contact to monopole antenna and the tube was ventilated from head to tail to decrease the stress of the rat while in the tube. The repetition time, frequency, and amplitude of the radio frequency energy spectrum were monitored by a satellite level meter (PROMAX, MC-877C, Barcelona, Spain).

Radiation reflection and exposure were measured with a Portable radio frequency Survey System (HOLADAY, HI-4417, Minnesota, USA) with a standard probe. The electromagnetic radiation dose was calculated from the measured electric field density (V/m). The whole-body

SAR values are in the range of 0.008–4.2 W/kg, representing a SAR value of 0.143 W/kg for whole body, with a value of 11.07 V/m at the closest point in the body.

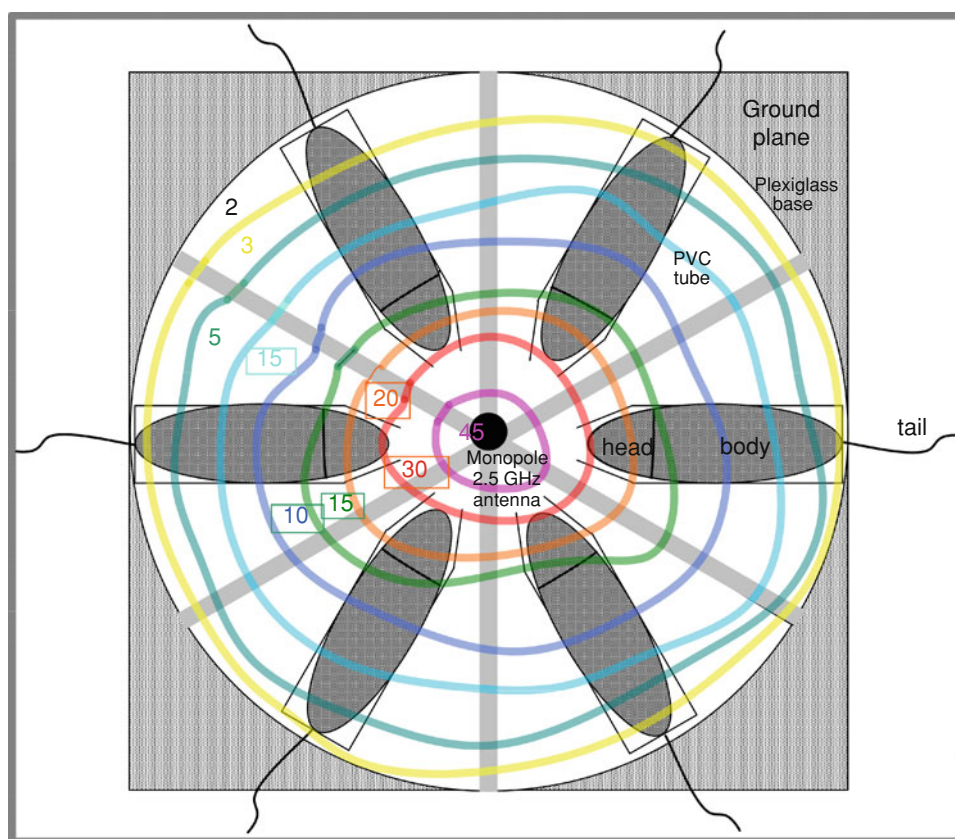
The whole exposure system was kept in a faraday cage with a shielding effectiveness of 100 dB. In each case, it was insured that the rest of the animals were not in any contact with the radiation-generating device by measuring the radio frequency.

The rats of sham control were placed in the cylindrical restrainer with the radio frequency source switched off during times similar to those used for irradiation. The cage control animals were kept in their cage without any treatment or restraint of any kind.

#### Preparation of laryngotracheal mucosa samples

Head of all animals were cut and the mucosal tissues were dissected from cartilages of larynx and trachea. The mucosal tissues were placed into glass bottles, labeled and stored in a deep freeze ( $-33^{\circ}\text{C}$ ) until processing

**Fig. 1** The experimental setup for irradiation of rats



Measuring parameters	Values
Power ( $\text{mW}/\text{m}^2$ )	1.0
SAR	0.1434
Distance between head of rat and antenna (m)	1.0

(maximum 10 h). After weighing, half of the mucosa were placed on ice and homogenized (2 min at 5,000 rpm) in five volumes (1:5, w/v) of ice-cold Tris–HCl buffer (50 mM, pH 7.4), using a glass Teflon homogenizer (Çalışkan Cam Teknik, Ankara, Turkey). All preparation procedures were performed on ice. After the addition of butylhydroxytoluol (4 µl/ml), mucosa homogenate samples were used for immediate lipid peroxidation, GSH levels and GSH-Px enzyme activities.

#### Determination of Lipid peroxidation level

Lipid peroxidation levels in the mucosa homogenate were measured with the thiobarbituric acid reaction by the method of Placer et al. [14]. The quantification of thiobarbituric acid reactive substances was determined by comparing the absorption to the standard curve of malondialdehyde equivalents generated by acid catalyzed hydrolysis of 1,1,3,3 tetramethoxypropane.

Laryngotracheal mucosa reduced glutathione (GSH), glutathione peroxidase (GSH-Px) and protein assays

The GSH content of the mucosa homogenate was measured at 412 nm using the method of Sedlak and Lindsay [15]. GSH-Px activities of the mucosa homogenate were measured spectrophotometrically at 37 °C and 412 nm according to the Lawrence and Burk [16]. The protein content in the mucosa homogenate was measured by method of Lowry et al. [17] with bovine serum albumin as the standard.

#### Statistical analyses

All results are expressed as mean  $\pm$  standard deviation (SD). To determine the effect of treatment, data were analyzed using analysis of Mann–Whitney *U* test. The *p* values of less than 0.05 were regarded as significant. Data were analyzed using the SPSS statistical program (version 17.0 software, SPSS Inc., Chicago, IL, USA).

## Results

The lipid peroxidation values in cage control, sham control, 2.45 GHz and 2.45 GHz + melatonin groups are shown in Fig. 2. Mean values as µmol/g protein in cage control, sham control, 2.45 GHz and 2.45 GHz + melatonin groups were 18.6, 18.6, 22.0 and 17.2, respectively. Lipid peroxidation levels were significantly ( $p < 0.05$ ) higher in 2.45 GHz group than in cage and sham control groups. However, melatonin induced lipid peroxidation lowering effects and lipid peroxidation levels and its level was

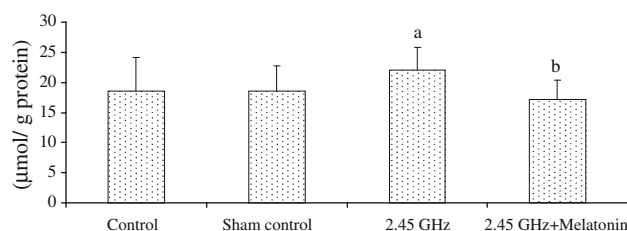
significantly ( $p < 0.01$ ) lower in 2.45 GHz + melatonin than in 2.45 GHz group.

GSH-Px activities in the four groups are shown in Fig. 3. Mean activities of GSH-Px as IU/g protein in cage control, sham control, 2.45 GHz and 2.45 GHz + melatonin groups were 23.4, 24.1, 23.1 and 30.4, respectively. GSH-Px activities were insignificantly lower in 2.45 GHz group than in cage and sham control groups, although its activity was significantly ( $p < 0.05$ ) higher in 2.45 GHz + melatonin group than in 2.45 GHz group.

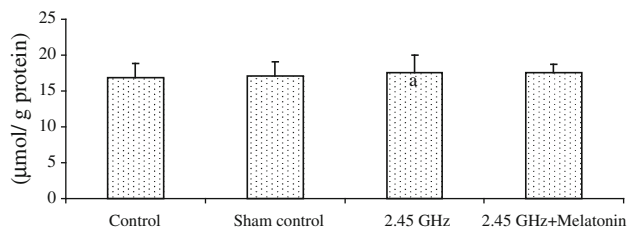
GSH levels in the four groups are shown in Fig. 4. Mean levels of GSH as µmol/g protein in cage control, sham control, 2.45 GHz and 2.45 GHz + melatonin groups were 16.8, 17.1, 17.6 and 17.5, respectively. There was no significant difference with respect to the levels of GSH among the groups.

## Discussion

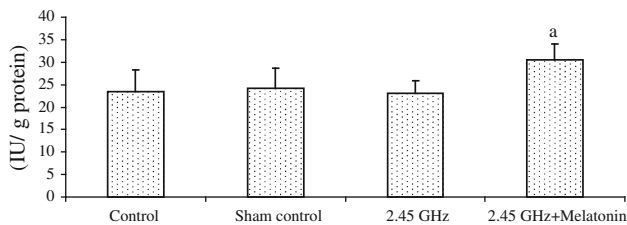
Lipid peroxidation is an oxidative stress marker and may trigger ROS-mediated tissue injury, such as MDA [18, 19]. It has been proposed that MDA acts as a tumor promoter and cocarcinogenic agent because of its high cytotoxicity and inhibitory action on protective enzymes [20]. Previous studies reported that plasma MDA levels were found to be significantly higher in the patients with laryngeal cancer than in the healthy controls [20, 21]. Furthermore, Inci et al. [22] reported that lipid peroxidation levels of cancerous tissues were found to be significantly higher than in the cancer-free adjacent tissues in the patients with laryngeal cancer. Previous studies have shown that 2.45 GHz EMR increased lipid peroxidation levels in various tissues including dorsal root ganglion neurons [11], plasma [4], erythrocytes [4], skin [23] and human leucemia 60 cancer cell line [8]. To our knowledge, this study is the first to report the effects of EMR on laryngotracheal mucosal tissues. The present study was shown that Wi-Fi (2.45 GHz) EMR (for 1 h/day for 30 days) increased lipid peroxidation levels in laryngotracheal mucosal tissues of rats. Hence,



**Fig. 2** Effects of melatonin on 2.45 GHz-induced lipid peroxidation levels in laryngotracheal mucosa of rat (mean  $\pm$  SD and  $n = 8$ ). <sup>a</sup> $p < 0.05$  versus control and sham control groups. <sup>b</sup> $p < 0.01$  versus 2.45 GHz group



**Fig. 3** Effects of melatonin on 2.45 GHz-induced reduced glutathione (GSH) levels in laryngotracheal mucosa of rat (mean  $\pm$  SD and  $n = 8$ )



**Fig. 4** Effects of melatonin on 2.45 GHz-induced glutathione peroxidase (GSH-Px) activity in laryngotracheal mucosa of rat (mean  $\pm$  SD and  $n = 8$ ). <sup>a</sup> $p < 0.05$  versus 2.45 GHz group

lipid peroxidation levels of the present study were confirmed by results of the recent studies [4, 8, 11, 23].

The GSH and GSH-dependent enzyme system also protect cells against ROS by scavenging hydro- and lipid peroxides [6]. GSH-Px is synthesized from GSH. Hydrogen peroxide is converted to water by catalase and GSH-Px. In the present study, we were not able to find statistical changes in GSH and GSH-Px values between 2.45 GHz and control groups. We were also not able to measure catalase values in the present study. We suppose that the ROS may be inhibited by catalase instead of GSH-Px. Hence, the GSH and GSH-Px values did not change in the present study between 2.45 GHz and control groups. Future studies should aim to measure the catalase activity in the 2.45 GHz-exposed laryngotracheal mucosa.

Contrary, Inci et al. [22] reported that GSH level and GSH-Px activity were higher in the laryngeal cancer tissue than in cancer-free adjacent tissues. Similarly, Kacakci et al. [21] observed no significant difference between the GSH levels of cancerous and cancer-free adjacent laryngeal tissues, whereas in blood, it was significantly higher in the control group, in their study. Ceyhan et al. [23] reported that EMR (2.45 GHz/h/day for 28 days) increased GSH-Px levels in skin tissues. Gümral et al. [4] reported that EMR (2.45 GHz/h/day for 28 days) decreased GSH-Px levels in erythrocytes. Similarly, Nazıroğlu and Gümral [24] and Nazıroğlu et al. [11] reported that EMR (2.45 GHz/h/day for 28 days) did not have effective

influence on GSH and GSH-Px levels in brain. We used similar setup and exposure time in the recent studies [4, 11, 23] and GSH and GSH-Px results of the present study were confirmed by reports of Kacakci et al. [21] and Nazıroğlu and Gümral [24] and Nazıroğlu et al. [11].

In results of the present study, lipid peroxidation levels were lower in 2.45 GHz + melatonin groups than in 2.45 GHz groups, although GSH-Px activities were higher in 2.45 GHz + melatonin groups than in 2.45 GHz groups. Melatonin, the main secretory neurohormone of the pineal gland, has been considered a potent anti-oxidant that detoxifies a variety of ROS in many pathophysiological states [9]. Pre-treatment with melatonin prevented the high lipid peroxidation level in the present study. All data reported above aid in identifying that melatonin may play an antioxidant role against EMR-induced oxidative injury. According to Kesari et al. [25], the 900 MHz EMR exposure produced an excess of ROS production according to recent data via the mitochondrial respiratory chain and impaired the antioxidant defense system. It has been hypothesized that melatonin, a lipophilic compound acts by a direct or indirect mechanism on ROS production [9]. It is known that melatonin can directly scavenge free radicals [26]. It has also been shown that melatonin has an antioxidant effect in other experimental models, such as dorsal root ganglion neuron [11] and hippocampus [27] and that it plays a protective role against EMR-induced neuron toxicity through its effect on mitochondrial chain, transient receptor potential melastatin 2 cation channels, and oxidative enzymes [11, 25].

In conclusion, in rats, exposure to 2.45 GHz EMR is accompanied by increased oxidative stress, suggesting that oxidative stress is a cause of EMR-induced laryngotracheal pathophysiology. Result of this study indicated that melatonin plays a protective action against 2.45 GHz EMR-induced oxidative stress in the laryngotracheal mucosa. The moderate melatonin supplementation may play a role in 2.45 GHz-induced laryngotracheal oxidative toxicity due to the exposure to EMR. The results may be useful in the etiology of electromagnetic radiation-induced larynx cancer. Nevertheless, more studies regarding the exact mechanism by which melatonin improves the EMR-induced oxidative stress in the larynx carcinoma are required to account for these data. However, results of the study are not relevant yet for clinical relevance. Future studies should therefore be aimed at identifying the specific intracellular pathways that transduce the Wi-Fi-induced changes in oxidative stress of exposed larynx of animal and human.

**Conflict of interest** None of the authors has any conflict of interest, financial or otherwise.

## References

- Öngel K, Gümrall N, Özgüner F (2010) The potential effects of electromagnetic field: a review. *Cell Membr Free Radic Res* 1:85–89
- Crouzier D, Testylier G, Perrin A et al (2007) Which neurophysiologic effects at low level 2.45 GHz RF exposure? *Pathol Biol (Paris)* 55:235–241
- Lukac N, Massanyi P, Roychoudhury S et al (2011) In vitro effects of radiofrequency electromagnetic waves on bovine spermatozoa motility. *J Environ Sci Health A Tox Hazard Subst Environ Eng* 46:1417–1423. doi:[10.1080/10934529.2011.607037](https://doi.org/10.1080/10934529.2011.607037)
- Gümrall N, Naziroglu M, Koyu A et al (2009) Effects of selenium and L-Carnitine on oxidative stress in blood of rat induced by 2.45-GHz radiation from wireless devices. *Biol Trace Elem Res* 132:153–163. doi:[10.1007/s12011-009-8372-3](https://doi.org/10.1007/s12011-009-8372-3)
- Türker Y, Naziroglu M, Gümrall N et al (2011) Selenium and L-carnitine reduce oxidative stress in the heart of rat induced by 2.45-GHz radiation from wireless devices. *Biol Trace Elem Res* 143:1640–1650. doi:[10.1007/s12011-011-8994-0](https://doi.org/10.1007/s12011-011-8994-0)
- Anderson ME (1998) Glutathione: an overview of biosynthesis and modulation. *Chem Biol Interac.* 111–112:1–14
- Naziroglu M (2009) Role of selenium on calcium signaling and oxidative stress- induced molecular pathways in epilepsy. *Neurochem Res* 34:2181–2191. doi:[10.1007/s11064-009-0015-8](https://doi.org/10.1007/s11064-009-0015-8)
- Naziroglu M, Çiğ B, Doğan S et al (2012) 2.45-Gz wireless devices induce oxidative stress and proliferation through cytosolic  $Ca^{2+}$  influx in human leukemia cancer cells. *Int J Radiat Biol* 88:449–456. doi:[10.3109/09553002.2012.682192](https://doi.org/10.3109/09553002.2012.682192)
- Tan DX, Manchester LC, Terron MP et al (2007) One molecule, many derivatives: a never-ending interaction of melatonin with reactive oxygen and nitrogen species? *J Pineal Res* 42:28–42. doi:[10.1111/j.1600-079X.2006.00407.x](https://doi.org/10.1111/j.1600-079X.2006.00407.x)
- Fic M, Podhorska-Okolow M, Dziegiel P et al (2007) Effect of melatonin on cytotoxicity of doxorubicin toward selected cell lines (human keratinocytes, lung cancer cell line A-549, laryngeal cancer cell line Hep-2). *In Vivo.* 21:513–518
- Naziroglu M, Çelik Ö, Özgül C et al (2012) Melatonin modulates wireless (2.45 GHz)-induced oxidative injury through TRPM2 and voltage gated  $Ca(2+)$  channels in brain and dorsal root ganglion in rat. *Physiol Behav* 105:683–692. doi:[10.1016/j.physbeh.2011.10.005](https://doi.org/10.1016/j.physbeh.2011.10.005)
- Karaman E, Uzun H, Papila I et al (2010) Serum paraoxonase activity and oxidative DNA damage in patients with laryngeal squamous cell carcinoma. *J Craniofac Surg.* 21:1745–1749. doi:[10.1097/SCS.0b013e3181f4040a](https://doi.org/10.1097/SCS.0b013e3181f4040a)
- Manjunath MK, Annam V, Suresh DR (2010) Significance of free radical injury in laryngeal and hypopharyngeal cancers. *J Laryngol Otol* 124:315–317. doi:[10.1017/S0022215109991721](https://doi.org/10.1017/S0022215109991721)
- Placer ZA, Cushman L, Johnson BC (1966) Estimation of products of lipid peroxidation (malonyldialdehyde) in biological fluids. *Anal Biochem* 16:359–364
- Sedlak J, Lindsay RHC (1968) Estimation of total, protein bound and non-protein sulfhydryl groups in tissue with Ellmann's reagent. *Anal Biochem* 25:192–205
- Lawrence RA, Burk RF (1976) Glutathione peroxidase activity in selenium-deficient rat liver. *Biochem Biophys Res Commun* 71:952–958
- Lowry OH, Rosebrough NJ, Farr AL et al (1951) Protein measurement with the Folin phenol reagent. *J Biol Chem* 193:265–275
- Kovacic P, Somanathan R (2008) Unifying mechanism for eye toxicity: electron transfer, reactive oxygen species, antioxidant benefits, cell signaling and cell Membranes. *Cell Membr Free Radic Res* 2:56–69. doi:[10.3109/10799890903582578](https://doi.org/10.3109/10799890903582578)
- Kumar S, Kesari KK, Behari J (2011) The therapeutic effect of a pulsed electromagnetic field on the reproductive patterns of male Wistar rats exposed to a 2.45-GHz microwave field. *Clinics (Sao Paulo)* 66:1237–1245
- Taysi S, Uslu C, Akcay F et al (2003) Malondialdehyde and nitric oxide levels in the plasma of patients with advanced laryngeal cancer. *Surg Today* 33:651–654
- Kacacki A, Aslan I, Toplan S et al (2009) Significance of the counteracting oxidative and antioxidative systems in the pathogenesis of laryngeal carcinoma. *J Otolaryngol Head Neck Surg.* 38:172–177
- Inci E, Civelek S, Seven A et al (2003) Laryngeal cancer: in relation to oxidative stress. *Tohoku J Exp Med* 200:17–23
- Ceyhan AM, Akkaya VB, Güleçol SC et al (2012) Protective effects of  $\beta$ -glucan against oxidative injury induced by 2.45-GHz electromagnetic radiation in the skin tissue of rats. *Arch Dermatol Res* 304:521–527. doi:[10.1007/s00403-012-1205-9](https://doi.org/10.1007/s00403-012-1205-9)
- Naziroglu M, Gümrall N (2009) Modulator effects of L-carnitine and selenium on wireless devices (2.45 GHz)-induced oxidative stress and electroencephalography records in brain of rat. *Int J Radiat Biol* 85:680–689. doi:[10.1080/09553000903009530](https://doi.org/10.1080/09553000903009530)
- Kesari KK, Kumar S, Behari J (2011) 900-MHz microwave radiation promotes oxidation in rat brain. *Electromagn Biol Med* 30:219–234. doi:[10.3109/15368378.2011.587930](https://doi.org/10.3109/15368378.2011.587930)
- Reiter RJ, Tan DX, Osuna C et al (2000) Actions of melatonin in the reduction of oxidative status. *J Biomed Sci* 7:444–458
- Köylü H, Mollaoglu H, Ozguner F et al (2006) Melatonin modulates 900 MHz microwave-induced lipid peroxidation changes in rat brain. *Toxicol Ind Health* 22:211–216